CURENT TRENDS OF PREVALENCE AND CLINICAL SPECTRUM OF PULMONARY TUBERCULOSIS IN DIABETES MELLITUS AND ITS RELATIONSHIP WITH GLYCEMIC STATUS

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CHENNAI

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF CURRENT TRENDS OF PREVALENCE AND CLINICAL SPECTRUM OF PULMONARY TUBERCULOSIS IN DIABETES MELLITUS AND ITS RELATIONSHIP WITH GLYCEMIC STATUS"** submitted by **Dr. A. PRAKASH,** to the Tamil Nadu Dr. M.G.R. Medical University Chennai is in partial fulfillment of the required of the award of M.D. DEGREE BRANCH –I (General Medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of the Unit Chief

Signature of Professor
And HOD.

Signature of Dean

DECLARATION

I Solemnly declare that the dissertation titled **"A STUDY OF CURRENT TRENDS OF PREVALENCE AND CLINICAL SPECTRUM OF PULMONARY TUBERCULOSIS IN DIABETES MELLITUS AND ITS RELATIONSHIP WITH GLYCEMIC STATUS"** was done by me at Stanley Medical College and Hospital during 2008-2010 under guidance and supervision of **Prof. G.SUNDARAMURTHY, M.D.,**

The dissertation of submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirement for the award of M.D. DEGREE (BRANCH-I) in General Medicine.

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INTRODUCTION

Tuberculosis is, one of the oldest disease known to affect humans, is a major cause of death worldwide. This is a bacterial infection, which is caused by mycobacterium tuberculosis . Usually affect the lungs, although other organs are involved in up to one-third of cases. If properly treated, tuberculosis caused by drug susceptible strains is curable in virtually all cases. Transmission usually take place through the airborne spread of droplet nuclei produced by patients with infectious pulmonary tuberculosis.

Diabetes and Pulmonary Tuberculosis :

A variety of diseases and conditions favour the development of active tuberculosis. Likewise relative risk of developing pulmonary tuberculosis of all types and bacteriologically confirmed cases were 4 to 5 times higher in the diabetic patients than the matched controls. So diabetes was associated with an increased risk of pulmonary tuberculosis. Henceforth patients with Diabetes mellitus may be important targets for pulmonary Tuberculosis.

As well, effort to diagnose, detect and treat, Diabetes mellitus may have a beneficial impact on pulmonary tuberculosis control.

Historical overview of the Tuberculosis

Gutierrez and her colleagues at the Pasteur institute have concluded that, the progenitor of mycobacterium tuberculosis emerged from an array of mycobacterial species about 3 million years ago, presumably infecting early hominids and other primates in prehistoric times. It seems likely that all modern members of the mycobacterium tuberculosis complex evolved from a common ancestor 15,000 – 20,000 years ago[10].

Tuberculosis is a disease that has afflicted human kind throughout recorded history. Spinal lesion that are highly suggestive of tuberculosis have been observed in a skeleton recovered from a grave near Heidelberg that dates from 5000 BC; a skeleton excavated from the arena candide cave in Liguria, Italy that dates from 4000 BC; and similarly ancient graves in Denmark and the Jordan valley. Mycobacterium tuberculosis DNA has been recovered from a pre-Columbian mummy from Peru[11].

The earliest archaeological evidence of human TB comes from Egyptian art and mummies, there is ample evidence of spinal TB (Pott's disease) as early as 5,500 years ago. There are unequivocal references to TB in the old testament books of Deuteronomy and Leviticus at the time when jews were in exile in Egypt.

There is General agreement that TB first appeared as a human disease in East Central Africa and that it travelled with early peoples as they migrated into Asia minor and across the globe. There are imprecise prehistoric references to TB from INDIA and CHINA, but no archaeological evidence.

Tuberculosis in India has worst scourge bordering on silent genocide. Despite the fact BCG vaccination is given at birth all over the country, TB remains the biggest killer disease in India. India bears 28.4% of the entire world's TB burden.

There have been references to TB in the Vedas and it was called "Rajayakshma".

In the Krishna Yajurveda Samhita, there is reference to how Soma (Moon) had been affected by 'Yakshma', since soma, who affected by yakshma' it came to be known as Rajayakshma – Vagabhata, Ast-s, and Ast-hrd, Nidana (Vol.1-2)[22].

The great Indian physician SUSRUTA in 600 A.D. while (Avicenna 780-1027 AD) had commented that phthisis frequently complicated diabetes.

In classical Greece, Hippocrates, Plato and Aristotle described TB, and the word "Phthisis" for pulmonary TB in Greek, which means to melt or waste

away. Aristotle noted its contagious nature, but it was not until Koch discovered the tubercle bacillus in 1882.

Phthisis makes its attacks chiefly between the age of eighteen and thirty five; Hippocrates wrote. Tuberculosis was described by GALAN, the Greek Physician who settled in Rome and became its preeminent physician. He Recommended Fresh air, milk and sea voyages for his TB patients, treatment modalities that would persist well into the nineteenth and twentieth centuries.

There is also abundant evidence that scrofula – TB of the Cervical lymph nodes- was common in mediaeval Europe. Beginning with the Frankish king clovis in 496, European rulers practiced healing of this affliction by the royal touch, and later rulers supported some of the large expenses of the throne by charging the thousands of supplicants who sought their cures.

Increasing urbanization led to rising TB incidences. St. Francis died of TB in 1226; Baruch Spinoza succumbed in 1677.

In 1660 JOHN BUNYON called TB "The captain among these men of Death". At the time, case rates in London probably reached 1000-1250/ 100,000/ year[4].

As the epidemic wave of TB crested in Europe and North America in the eighteenth and nineteenth centuries, the disease became Romanticized.

Charles Dickens wrote, “There is a dread disease…. In which the struggle between body and soul is so gradual, quiet, and solemn…. (that as) the mortal part wastes and wither away, so the spirot grows light and sanguine[5].

Although TB had reached most of the globe in prehistoric times, it had long since receded from the Americas and much of Africa, leaving the peoples of those regions immunologically naive with Respect to Mycobacterium Tuberculosis.

Tuberculosis crested in Europe and North America at the middle of the nineteenth century and has been falling since that time to present historic low case rate.

Thomas Mckeown, a highly Respected population scientist, examined many possible causes for this decline and concluded that better nutrition played the major role.

Leonard Wilson argued that removal of infectious individuals to treatment facilities was a major factor. E.R.N. Grigg concluded that genetic herd immunity which developed as a Result of selection influenced by the high death rates in young TB adults resulted in the falling incidence[10].

Discovery of the causes :

In 1720 Benjamin marten published a remarkably clairvoyant book entitled, A new theory of consumption; More Especially of the phthisis or consumption of the lungs. The original and Essential cause, then wrote, may possibly be some certain species of animalcula or wonderful minute living creatures[9].

Jean – Antoine Villemin , a French military surgeon, who convincingly demonstrated the infectious nature of TB.

The Great pioneer of bacteriology ROBERT KOCH, demonstrated conclusively that a bacterium, which he called Bacillus Tuberculosis and is now known as Mycobacterium tuberculosis is the etiological agent of TB. His dramatic presentation of his findings on 24 March 1882 was immediately hailed, and he was acclaimed throughout the world.

Diagnosis :

Koch's studies of TB led to his report of tuberculin, as noted below. Viennese paediatrician Clemens Freiherr von pirquet, one of the Founders of the Science of allergy, developed the intracutaneous tuberculin test much as we know it today[2].

The Further work of Florence seibert and others in the 1930s led to the production of tuberculin – purified protein derivative, the antigen used today for tuberculin skin testing.

In November 1895, Conrad wilhelm Rontgen, a distinguished physicist interested in Radiation, discovered X-rays. With in a month he had imaged his wife's hand. The American inventor, Thomas Alva Edison developed the first practical fluoroscope, making clinical Radiology a practical reality.

Francis Williams Reported on the Fluoroscopic examination of the lungs in more than 100 patients at the Boston city Hospital. During the second world war , France, the united state, and Germany introduced Radiographic Screening of military Recruits for TB.

The Search for a cure :

Koch, produce, tuberculin, a concentrated bacteria free, filtrate of liquid cultures of mycobacterium tuberculosis; it became of great importance in the diagnosis of tuberculosis infection.

None of these measures benefitted the dying poet john keats, however, Herman Brehmer opened the first sanatorium for TB patients in Goerbersdorf in the silesan Mountains of Prussia in 1859.

As early as 1696, Giorgio Baglivi observed that a patient with pulmonary TB improved after a sword wound Resulted in a pneumothorax. Pneumothorax was first introduced for therapy by Carlo Forlanini in 1894 with beneficial Results.

The procedure was Rapidly accepted and widely used to collapse pulmonary cavities during the next 50 years. Pneumoperitoneum was used for lower lobe cavities. About the same time, thoracoplasty came into use in those cases in which satisfactory collapse could not be achieved by pneumothorax.

In the late 1800s, before chemotherapeutic agents had been discovered, patients with tuberculosis were isolated in sanatoriums for nutrition and rest. Lung collapse therapy was performed by pneumothorax or various surgical techniques that frequently left patients disfigured for life.

Chemotherapy:

The idea that it might be possible to find an agent that was specifically curative was espoused by paul Ehrlich, who heard Koch's presentation. His work led to treatment for trypanosomiasis and syphilis.

In 1932, Gerhardt Domagk, a pathologist working at the laboratories of Bayer, the German chemical company discovered prontosil, the first sulphonamide, Domagk continued to work, and his efforts led to thioacetazone, the first mycobacteriostatic drug. Shortly there after Jorgen Lehman in Sweden discovered Paraamino Salicylic acid (PAS) also mycobacteriostatic. Selman Waksman with his junior colleagues Albert schatz and Elizabeth Bugie discovered streptomycin in 1943, the first mycobacteriocidal drug and an early member of a class of drugs that Waksman called Antibiotics.

Isoniazid was discovered almost simultaneously by three pharmaceutical companies in 1952 and Rifampicin in 1963.

Thus, in the 60 years since the introduction of the first effective drugs for the treatment of tuberculosis, the care of these patients has been transformed from long hospitalization and a lifetime of fear of relapse to a relatively short outpatient treatment regimen and cure.

The challenge today is not so much how to treat tuberculosis as how to ensure that patients receive the proper treatment. Failure to deliver proper care is the major cause for the development of drug resistant strains and the feat for the recrudescence of this dreaded malady. Thus, directly observed therapy i.e., DOTS has been adopted as the preferred method for insuring completions of effective therapy.

Prevention and Control :

With knowledge of Edward Jenner's Vaccinia prevention of small pox and Louiz pasteur's immunization treatment of Rabies, Albert Calmette decided to turn his effort at the Pasteur institute in Lille, France to developing a vaccine agent.

In 1921, Calmette, now in paris, was Ready to try the vaccine known as Bacillus calmette Guerin (BCG) in a human subject.

Current control strategies in most nations emphasize treatment under direct observation using optimal drug Regimens to reduce the further spread of tuberculosis bacilli. Despite the discovery of the cause of the tuberculosis, and the development of effective chemotherapy in the 1950s, tuberculosis continues to be a major pulmonary pathogen and is a leading cause of infectious death in adults worldwide.

AIM OF THE STUDY

1. To study the current prevalence of pulmonary tuberculosis in diabetic patients.

2. To study the spectrum of its distribution in terms of sex and age.

3. To study the symptomatology of subjects with diabetes alone and with pulmonary tuberculosis.

4. To study the hematological, biochemical and Radiological changes in patients with diabetes mellitus and pulmonary tuberculosis.

5. To study the pulmonary tuberculosis relationship with glycemic status.

REVIEW OF THE LITERATURE

The lung is the most commonly affected organ in tuberculosis. In the 2004 CDC surveillance reports, 79.5% of the newly diagnosed cases of tuberculosis had lung involvement. Similar rates of pulmonary involvement are found in both immunocompetent and immunocompromised hosts. Such as those with Human Immuno-deficiency Virus infection[7].

Patients with Diabetes mellitus are at increased risk of developing active TB, but this risk, which may be fivefold or more, varies according to the background prevalence of TB and various host factors, including the patients age and sex, body mass, duration of diabetes and most importantly, adequacy of glycemic control.

The worldwide prevalence of diabetes has raisen dramatically over the past 2 decades, from an estimated 30 million cases in 1985 to 177 million in 2000. Based on current trends, more than 360 million individuals will have diabetes by the year of 2030. The prevalence of Type 2 diabetes is rising much more rapidly because of increasing obesity and reduced activity levels as countries become more industrialized.

The prevalence of type 2 diabetes and its harbinger, IGT, is highest in certain pacific islands, intermediate in countries such as INDIA and the United

States and relatively low in Russia. A recent estimate suggested that diabetes was the 5th leading cause of death worldwide, and was responsible for almost 3 million deaths annually (1.72-5.2% of death worldwide).

Individuals with diabetes mellitus have a great frequency and severity of infection. The reasons for these include incompletely defined abnormalities in cell mediated immunity and phagocytes function associated with hyperglycemia, as well as diminished vascularization. So, a high prevalence of pulmonary tuberculosis was found in diabetic patients. This pulmonary tuberculosis is due to mycobacterium tuberculosis, is a rod-shaped, non-spore forming, thin aerobic bacterium measuring 0.5 micrometer/3 micrometer. Mycobacteria, including Mycobacterium Tuberculosis, are often neutral on gram's staining. However, once stained, the bacilli cannot be decolorized by acid alcohol; this characteristic justifies there classification as Acid-Fast Bacilli.

More than 5 million new cases of tuberculosis (all forms, both pulmonary and extra-pulmonary) were reported to the WHO in 2005; more than 90% of cases were reported from the developing countries. However, because of insufficient case detection and incomplete notification, reported cases represent only approximately 60% of the total estimated cases.

[12]The WHO estimated that 8.8 million new cases of tuberculosis occurred worldwide in 2005, 95% of them in developing countries of Asia (4.9 million);

Africa (2.6 million); the middle-east (0.6 million) and Latin America (0.4 million). It is further estimated that 1.6 million deaths from tuberculosis occurred in 2005, 95% of them in developing countries. Estimates of tuberculosis incidence rates and number of deaths due to tuberculosis in 2005 in India is 100-299/100,000 population and more than 100,000, respectively.

In 1883, Windle autopsied 333 known diabetic subjects and observed pulmonary tuberculosis, in more than 50% of them. In a classic study, Root Reported that 2.8% of 1373 hospitalized diabetics had pulmonary Tuberculosis of the 750 juvenile diabetics, 1.6% had tuberculosis as compared to 0.12% among school children. After studying the association between Diabetes and Tuberculosis, he made the following observation[2].

i) The development of TB occured ten times more frequently in juvenile diabetics.

ii) In 85% of the patients, Tuberculosis had developed after the onset of Diabetes.

iii) The occurence of pulmonary Tuberculosis increased with the duration of Diabetes.

[3]The Philadelphia survey revealed that 8.4% of the 3,106 diabetes had pulmonary tuberculosis as compared to 4.3% of the 71,767 presumably healthy

industrial workers. TB was present in 17% of the DM who had had the disease for more than 10 yrs. Compared to 5% in DM with less than 10 yrs of the disease.

A higher prevalence of TB was found in diabetes patients requiring more than 40 units of insulin per day.

In countries like INDIA, diabetes remains one of the most important Risk factor predisposing towards tuberculosis, along with malnutrition, alcoholism and HIV infection. The prevalence of pulmonary tuberculosis in diabetics in INDIA varies from 3.3% to 8.3% about 4 times that of General population[6].

The purported relationship between DM and TB dates back to Roman times. In an extensive Recent Review of the epidemiology of TB Rieder et al. have drawn as 3 larger survey's from 1950s. It suggest relative risk of TB in individuals with diabetes that is 2-3.6 times that in those of non DM.

Factors influencing Susceptibility to Tuberculosis in diabetic patients:

i) Age and Duration of diabetes :

a. Diabetes over the age of 40 years are at increased risk of developing TB and a longer duration of diabetic disease predisposes to TB in all age group[19].

ii) Sex :

a. Male diabetics are generally considered to be at greater risk of TB than female.

iii) Glycemic Control :

Increased risk of smear positive disease has been demonstrated at Hb A1c levels of 9% or more.

iv) Body Mass :

Low body mass is a independent risk factor for TB.

Immune dysfunction in diabetes[17] :

A probable cause of increased incidence of pulmonary Tuberculosis in DM could be defect in host defenses and immune cell function.

The immune derangements predominantly involve the cell mediated immunity arm of the immune system. Also the degree of Hyperglycemia has been found to have a district influence on the microbicidal function of macrophages, with even brief exposures to blood sugar level of 200mg% significantly depressing the Respiratory burst of these cells.

Multiple pulmonary physiologic abnormalities have also have also been documented in diabetics that contributes to delayed clearance of the spread of infection in the host infection with Tubercle bacilli leads to further alteration in cytokines, monocytes , macrophages and CD4/CD8 T cell populations. The balance of the T-lymphocyte subset CD4 and CD8 plays a central role in the modulation of host defenses against mycobacteria and has a profound influence on the rate of regression of active pulmonary tuberculosis.

List of defect in diabetics immunologic make up and physiologic pulmonary functions.

Immunologic abnormalities in diabetes	**Pulmonary Physiologic dysfunction in diabetes.**
Abnormal Chemotaxis adherence, phagocytosis and microbicidal function of polymorph.	Diminished bronchial reactivity.
Decreased Peripheral monocytes with impaired phagocytes.	Reduced elastic Recoil and lung volumes.
Poor blast transformation of lymphocytes.	Reduced diffusion capacity.
Defective C_3 opsonic function.	Reduced ventilator Response to Hypoxemia

Cellular immunity :

The levels of interferon gamma and interleukin 12 productions in diabetic patients were significantly lowered. These cytokines production were significantly reduced in patient with TB with DM than TB alone.

The levels of IL-12 in tuberculous patients were highest. But the patients cytokines in TB and DM was lowest. The low interferon gamma correlates with DM control. The period for Negative control finding in TB and DM patients under poor control were compared with those with TB alone[18].

Cellular immunity of Pulmonary Tuberculosis in patients with Diabetes. (Russian-Rroblemy – 1997)

The parameters of cellular immunity were studied in 64 patients with pulmonary tuberculosis developed in the presence of Type I DM compared with the patients with pulmonary TB alone.

Patients with concomitant abnormalities showed higher depression of cellular immunity appeared as fewer T-lymphocytes are decreased capacity for blast cell transformation than those with TB alone[11].

Several groups of investigators have found patients with diabetes mellitus to be more susceptible to tuberculosis, the overall increased risk being in the order of 2 to 3.5. Some have also documented an increased likelihood of lower lobe or multiple lobe disease, pleural effusion and cavitations.

Numerous Risk factors have been indentified; among the more important ones are diabetes, silicosis, low body weight and smoking.

A variety of disease conditions favour the development of active tuberculosis. These are HIV, DM and Silicosis, CRF etc[14].

In General, the organisms, that causes pulmonary infections are similar to those found in non diabetic population, however, gram Negative organisms, staphylococcus aureus and mycobacterium Tuberculosis are more frequent pathogens in DM[12].

Pulmonary Complication of DM[17]

The lung is not considered a target organ in diabetes mellitus. Many model demonstrated.

1. Relative paucity of physical signs which may delay the diagnosis of TB in DM patients.
2. Extensive caseation of lung tissue and cavitary lesion is more common.
3. Middle and lower lobe predilection probably due to bronchial lymph node involvement.
4. Limited pleural involvement.
5. Greater tendency of hemoptysis.
6. Less chance of extrapulmonary involvement.
7. More cases of sputum +ve disease.

Clinical Features

The clinical features and presentation of TB in most patients with diabetes are similar to those without, but unusual presentation haves been described. Higher incidence of Extrapulmonary tuberculosis and certain rare manifestations are higher than in normal persons, like laryngeal TB.

The most frequently reported symptoms in studies of active pulmonary tuberculosis include cough (23-47%), fever (18-79%), weight loss (7-24%) and hemoptysis (8-9%).

Symptoms of one disease often mimic those of the other. Loss of weight, Loss of appetite and Lassitude are common to both the disease.

An overview of Radiological lesion peculiarity in patients with combined diabetes and pulmonary Tuberculosis :

Sosman and steidl reported that diabetic tuberculosis has a specific Radiological pattern consisting of confluent, cavitary, wedge shaped lesion spreading from the hilus towards the periphery, predominantly in lower zone, to the extent of around 20%.

Marias observed lower lung field tuberculosis in 29% of patients with DM, as compared to 4.5% in the non DM patients. However; in other studies, cavitary disease and multiple lobe involvement was found to be more common in patients with pulmonary tuberculosis with DM.

[3]Lower lobe pulmonary TB is not uncommon and the report appeared from literature from time to time. The earliest report are those of KIDO in 1886 and FOWLER in 1888, who noted the vulnerability of the spaces of lower lobe with the evidence of postero-anterior and lateral skiagram of the chest.

Management of Tuberculosis in DM patient[1] :

1. Proper Care.

2. Patient with poor diabetic control should be hospitalized for stabilizing their blood sugar level.

3. Ideally, insulin should be used to control blood sugar level.

4. OHA should be used only in cases of mild diabetes. Drug interaction with Rifampicin should be kept in mind.

5. Glycemic equilibrium is essential for the success of ATT and must be achieved in every patient with co-existent disease. The goal of therapy are : fasting plasma gluscose < 120 mgs and glycated Hb% <7%.

6. Vigorous and good chemotherapy is essential, monitoring for adverse effect, particularly of hepatic and nervous system should be done. Use of potentially Neuropathic agent (INH) in patients with peripheral Neuropathy demands special consideration with mandatory administration of pyridoxine.

7. Duration of chemotherapy is entirely dependent up on the control of diabetes and responses of the patient to treatment. A longer treatment course may be needed.

8. Supportive therapy for diabetes must be actively pursued.

9. Management of co-existent illnesses, malnutrition and rehabilitation of the alcoholic diabetics remain of prime concern.

10. If the DM is well controlled, response to ATT is same as non diabetic groups. However, if not under strict control, treatment of TB is often ineffective. Patients with type II DM on oral therapy may need of supplementation of insulin replacement. Type I patients may require intensified insulin treatment. Adequate insulination is important as it .

    a. Restore host defense response to infection.

    b. Restore anabolic process.

    c. Restore appetite and thereby improve weight.

    d. Restore the sense of well being[6].

    Many ATT drugs are metabolized in the liver. Rifampicin is crucial for the success of ATT regimen, it induce hepatic enzyme and inactivate sulphonylureas, metabolized by the liver, these drugs may become less effective and should preferably be avoided.

Some ATT have metabolically affect the endocrine affect of endocrine effect of DM, which can affect the diabetic status eg. Pyrazinamide. Rifampicin can impair Renal function and cause acute Renal failure even within few weeks of starting therapy. INH & Rifampicin both lead to abnormal Vit. D metabolism and hypocalcemia. PAS therapy is associated with Hypoglycemia.

The Recommendation for treatment of TB with DM patients ie. Four drug regimen (Rifampicin / INH/ Ethambutol / Pyrazinamicle) for 2 month intensive phase followed by 4 months of continuation phase. Patient should be monitored carefully, because danger of drug interaction. Based on clinical experience some patient not response to usual 6 months Regimen, they should be prolonged to nine months or even one year Regimen.

Prophylaxis

All diabetics required regular medical examination and bi-annual chest radiograph. This should be followed more vigorously in patients who are more than 40 yrs of age or with weight less than 10% of the ideal body weight.

Any diabetic –who suddenly develops cough, loss of weight, abnormal chest radiograph or needs increasing dose of insulin to control blood glucose should be investigated for presence of TB.

SUBJECT AND METHODS

A total of 100 cases of Diabetic patients attending medicine OPD/IPD who has chest symptoms of duration ranging from 3wks to 6 months with age group ranging from 18 years to 79 years of both male and females were selected.

Screening for pulmonary tuberculosis is to be done on these patients.

Place of Study

Out patients & Inpatients department

Department of Medicine

Stanley Medical College,

Chennai – 1.

Period of Study :

April 2010 – November 2010.

Inclusion Criteria

Adult patients ranging from 18 years to 79 years of age of both sex with Diabetes mellitus on Treatment

Exclusion Criteria

1. Patients with previous history of pulmonary Tuberculosis.
2. Patients with previous history of Extra Pulmonary Tuberculosis and exposure to ATT.

Study Method :

Prospective observational Study.

Subjects :

A total of 100 known diabetic patients, taking treatment in medicine department, who have chest symptoms of duration ranging from 3 weeks to 6 months with age group ranging from 18 to 79 years of both sexes were selected. They underwent screening for pulmonary Tuberculosis.

The study was carried out at Govt. Stanley Medical College Hospital, Medicine department with the active co-ordination and participation of chest clinic and other departments from April 2010 to November 2010. For the purpose of the study of known diabetic patient getting treatment in our hospital with chest symptoms like cough with expectoration (>3 weeks), chest pain , hemoptysis, fever were screened for the occurrence of TB, who have not been previously investigated.

The methodology comprised of history of symptomatology of TB, physical examination and laboratory investigation of Urine and Blood analysis for glucose, blood count, ESR, X-ray chest, Sputum AFB and mantoux.

Particulars of subjects with Diabetes alone and combination of Diabetes with pulmonary Tuberculosis - Number and Sex Ratio.

Number of Subjects	**Diabetes**	**Diabetes with Pulmonary TB**
	74	26
Sex	37	20
Male		
Female	37	6
Ratio M :F	1:1	3:1

History :

Enquiry of the subjects based on symptomatology, detailed history including the duration of both DM and TB. The average duration of the diabetes prior to the onset of TB varied between sexes. The duration of DM among the patients, who had pulmonary TB ranges from minimum of 1 year to maximum of 20 years.

Symptomatology;

Subjects with DM and also with a combination of DM and pulmonary TB were presented with the following chest symptoms of duration ranging from 3 weeks to 6 months. Underwent the investigation for confirming the diagnosis of pulmonary TB.

Details of Symptomatology investigated for TB:

Chest Symptoms presented in the study groups.

Cough	Chest pain
Hemoptysis	Loss of appetite and weight
Fever	Others.

Physical Examination

All the patient with established diabetes mellitus underwent physical examination which comprised, General Examination, Respiratory system examination and other system examination.

Particulars of physical examination of subjects.

Nature of Examination	Particulars
General Examination	Pulse, Blood Pressure, Temperature Built of individual, Height, Weight Body Mass index (BMI), Pallor, Clubbing, lymph node, cyanosis, leg edema.
Respiratory System Examination	Upper respiratory tract, Trachea, Chest deformity, Respiratory Movement, vocal fremitus, percussion, Auscultation, vocal resonance etc.
Other system examination	Cardiovascular, Abdomen and Nervous System.

Mantoux test :

This test was done by using purified protein derivative (PPD) RT 23 of one Tuberculin unit, injected on the volar aspect of the left forearm intradermally. Result was observed after 48 to 72 hours. As indurations of diameter 10mm or more was considered to positive.

X-ray Chest :

X-Ray chest Postero – Anterior view was taken for all patients in the study groups and lateral view was taken in selected cases. Number of Zones involved, nature of the lesion like infiltration, cavitation, consolidation and fibrocavity was documented.

The upper zone corresponds to an area between the apex of the lung and to a line drawn at a Lower border of second costal cartilage. The middle zone in between upper zone and fourth costal cartilage. Below the middle zone is the lower zone. Depending on the radiological lesion, pulmonary TB can be divided radiologically.

1. Early tubercular infiltration..
2. Pneumonic or broncho pneumonic[consolidation] lesions
3. Cavitary lesions.
4. Disseminated lesions.
5. Miliary lesions.

Study Results and Discussion

The results of the investigation are classified under following headings for the purpose of discussion.

A. Subject under study :

The study group comprised of 100 subjects who had established diabetes mellitus taking treatment at medicine department presented with the complaints of chest symptoms underwent screening for evidence of pulmonary TB. Out of 100 subjects 26 were found to be suffering from pulmonary TB. The Remaining 74 subjects had diabetic alone who responded for routine antibiotics for their chest symptoms. The spectrum of distribution in terms of sex and age groups are presented here. (Table-1).

Table - 1

Number, Sex and age group Breakup of subjects with combination of Diabetes and pulmonary Tuberculosis against Diabetes alone.

Number of Subjects	**Diabetes**	**Diabetes with Pulmonary TB**
	74	26
Sex	37	20
Male		
Female	37	6
M :F Ratio	1:1	3.3:1

	Diabetes alone			TB with DM		
Age Group	**Male**	**Female**	**Total**	**Male**	**Female**	**Total**
Below 9	-	-	-	-	-	-
10 -19	-	1	1	-	-	-
20-29	-	-	-	-	-	-
30-39	2	0	2	1	0	1
40-49	4	8	12	2	0	2
50-59	11	10	21	5	2	7
60-69	12	11	23	8	3	11
70-79	8	7	15	4	1	5
Total	37	37	74	20	6	26

Here incidence as well as prevalence are same in this study. The subjects studied for the incidence / prevalence of pulmonary TB among 100 diabetes patients, 26% were with evidence of pulmonary Tuberculosis, with Respect to sex, male account for three times that of female , where as the sex ratio is about equal in diabetes alone. This difference, suggests that, male are expected to have a greater risk to TB infection than females. Even though the number of subjects are enough to purpose a generalization. The causes may be many and also varied. Considering the economic strategic of the subjects, it may be associated under an environment peculiar to industrial area.

Age group break up presents a pattern, in which difference between the combination of TB with DM and diabetes alone can be made out, for instance the frequency diagram shows a sharp, peak for the age group of 60-70 years and the

high valve and have a narrow range in either side is 30-40 years and 70 years and above for cases of pulmonary TB with diabetes. But in the diabetes alone, patients present a frequency diagram entirely different. The trend is conspicuous by the absence of any high peak, a plateau of value can be noted widening the age group ranges between 40 years to 80 years. This at once suggests that the susceptibility is high in increased age in duration of diabetes.

B. Symptomatology:

In term of symptomatology, the subject suffering with diabetes and pulmonary TB and diabetes alone may be subjects symptoms wise, presented in table-2 .

Polyuria and polydypsia are quite common symptoms of Diabetes. Among the study group populations with diabetes alone, and combination with pulmonary Tb, the symptoms are presented in table-2.

Category of subjects	Polyuria	Polydypsia	Neuropathy	Pruritus	Cough with expecto ration	Hemoptysis	Fever	Chest pain	Loss of appet ite	Loss of weight	Others
Diabetes alone Total No; 74	21	17	14	1	74	0	19	10	07	0	2
% of Symptoms	28.37	22.97	18.91	1.35	100%	0	25.67	13.51	9.45	0	2.70
DM with Pul TB Total No. 26	20	15	16	2	26	7	15	4	14	4	2
% of symptoms	76.92	57.69	61.53	7.69	100%	26.92	57.69	15.38	53.84	15.38	7.69

Analyzing the symptomatology of the study group of diabetes with TB, apartfrom the symptoms of diabetes alone, these subjects presented with cough, fever, chest pain, hemoptysis, loss of appetite and of loss of weight with a percentage of 100, 57, 15, 26, 53 and 15 respectively. In contract to diabetes alone group, subjects with TB had fever and hemoptysis. The hemoptysis account 26% where as it is zero in diabetes alone, similarly loss of appetite and loss of weight account for 53% and 15% respectively, loss of weight is not a feature in diabetic alone and loss of appetite is not so high in diabetic alone patients. Any patients with established diabetes who complaints of loss of appetite and loss of weight carries significance and investigation is to be carried out to rule out Tuberculosis.

C.BLOOD COUNT DATA;

Blood count data – total and differential counts are comparing between combination of DM with pulmonary TB and Diabetes alone(Table-3,4).

Table -3

Total Counts and ranges in subjects with DM and pulmonary TB as against Diabetes alone.

Disease	Total Leucocytes count cells/cumm	Range Cells / cumm
DM with Pulmonary TB	6878	4200-11300
Diabetes alone	6337	4200-11500

Table -4

Differential Counts and range in subjects with Diabetes and Pulmonary TB as against Diabetes alone.

	Differential Counts and range		
	Polymorphs %	Lymphocytes %	Eosinophils / monocytes%
DM and Pulmonary TB	66 (55-82)	33 (24-45)	2 (0-10)
DM alone	68 (50-82)	31 (15-48)	2 (0-10)

Blood Glucose values as also insulin requirements and duration of diabetes with pulmonary TB and Diabetes alone (Table-5).

Table -5

Disease	Sex	Bl. Glucose mgs% F-fasting/PP-post prandial	Insulin requirement (Units)	Duration of diabetes (years)
Diabetes with Pulmonary. TB	Male	(F) – 283 PP – 387	40	7.4
M : 7 F : 4	Female	(F) - 298 PP – 387	39	8.0
Total No: 11	Mean	(F) - 291 PP – 387	40	7.7
Diabetes alone	Male	(F) – 137 PP – 219	21	3.6
M : 5 F : 7	Female	(F) - 139 PP – 211	19	3.9
Total No : 12	Mean	(F) - 138 PP – 215	20	3.75

D. Erythrocyte Sedimentation Rate :

ESR values are assessed. The ranges estimated between 12 to 40 a mean value of 35 mm/hr in the cases of diabetes with pulmonary TB. The range of ESR value among diabetes alone study group extended between 5 and 40 mm/hr, with a mean value of 16mm/hr. though the value among combination of diabetes with pulmonary TB ranges high, this against a non specific criteria and as in the previous cases serves a 60/hr as a diagnostic tool.

Table -6

Table showing blood glucose values and duration of Diabetes subjects on oral Hypoglycemic agents with pulmonary Tuberculosis and diabetes alone.

Disease	Sex	Bl. Glucose (mgs%)	Duration of Dm (yrs)
Diabetes with Pul TB	Male	F – 232 PP – 326	9.5
M : 13 F :2	Female	F – 168 PP – 314	9.5
Total No : 15	Mean	F – 200 PP – 320	9.5
Diabetes alone	Male	F – 141 PP – 216	4.7
M : 32 F :30	Female	F – 136 PP – 212	5.4
Total No: 62	Mean	F – 138 PP – 214	5.05

E. Sputum Examination for Acid Fast bacilli

All 100 subjects screened for pulmonary TB were subjected to sputum Examination for Acid Fast Bacilli. Out of 100 subjects, 12 subjects were found to be positive for Acid Fast Bacilli. Out of which, 8 were males and 4 were females. These subjects were subjected to sputum examination for three times, Two spot specimen and one overnight collection.

Table -7

Showing details of sputum Examination for AFB

Aspects	**MALE**								**FEMALE**			
Sputum for AFB-Serial	A	B	C	D	E	F	G	H	1	J	K	L
Sputum for AFB	+	+	+	+	+	+	+	+	+	+	+	+
Bl. Glucose	465	300	317	400	330	296	323	292	364	282	305	465
Insulin Requirement	45	40	35	45	45	36	38	36	45	36	45	45
Age (years)	51	49	75	45	60	65	64	77	62	64	65	71

Out of 12 sputum positive subjects, 11 were on insulin treatment, and remaining one patient, who was on oral Hypoglycemic agent treatment, also currently changed over to insulin.

Among the sputum positive patients **two** had Radiologically lower lobe involvement with diffuse lesion, **one** had consolidation, **five** had cavitary lesion with surrounding infiltration**, Four** had infiltrative lesion. Most of all, are multiple zonal involvement, only one had left upper zone involvement. Radiological lesions correlated with uncontrolled hyperglycemia and sputum positivity with severity of clinical signs.

F. Mantoux test :

Table -8

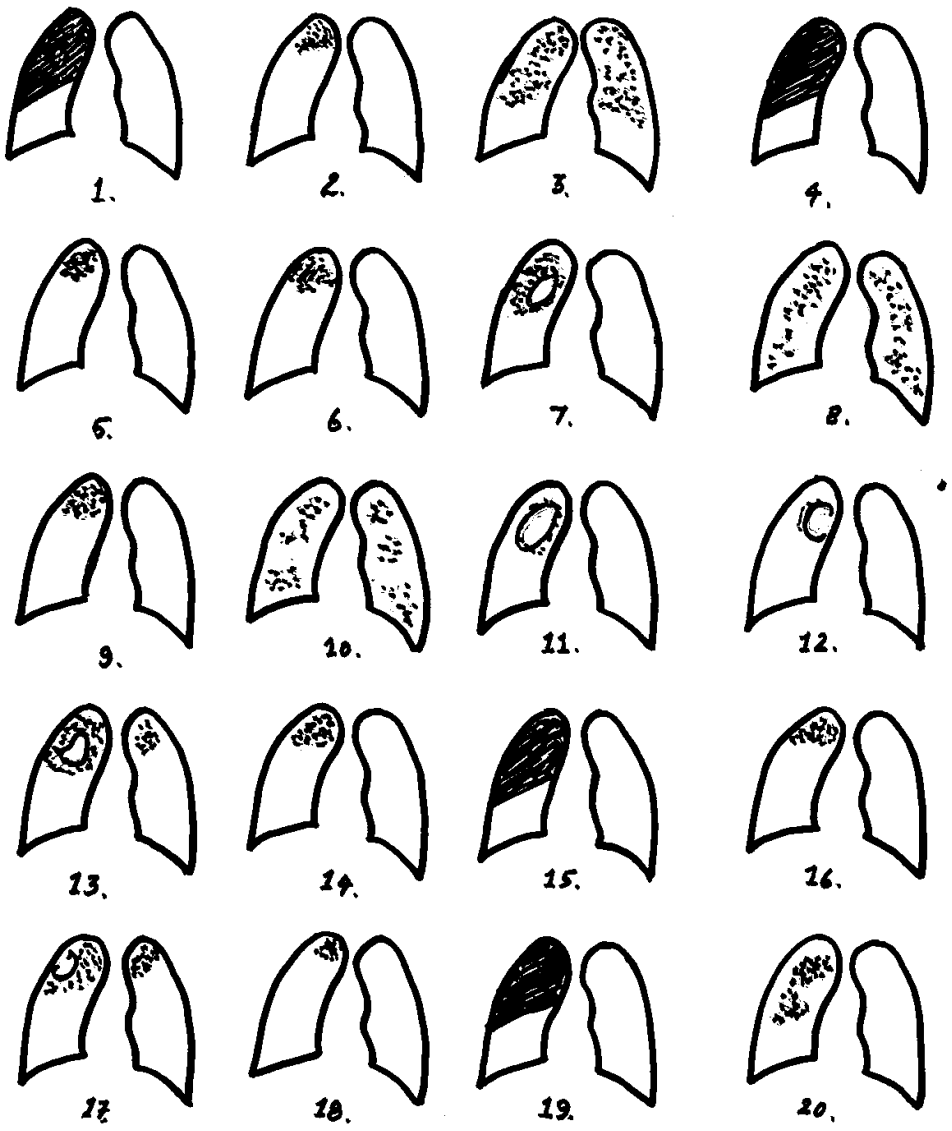
Table showing Results of Mantoux test

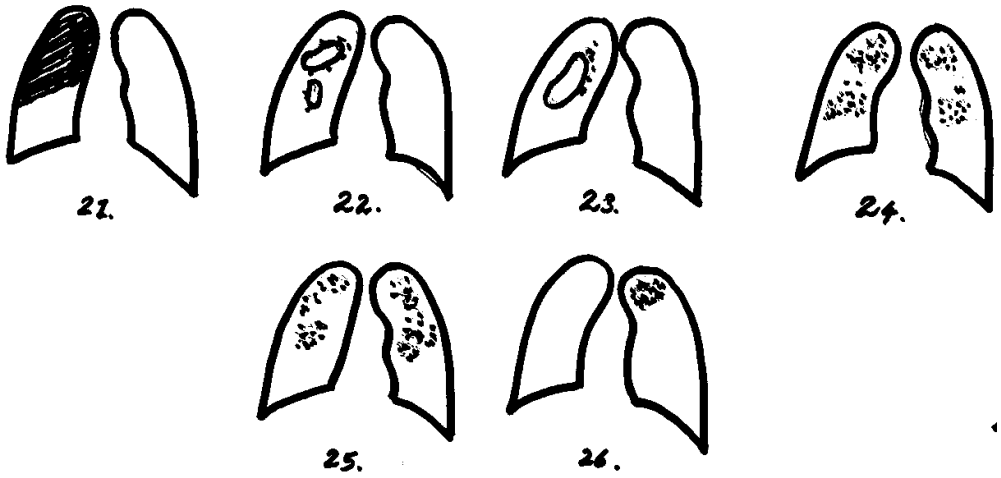
Male		**Female**	
MX +ve	Mx –ve	MX +ve	Mx –ve
16	4	4	2

The Mantoux test results shows, 20 subjects were showed positive Mantoux, 6 were negatives. All positive Mantoux subjects had pulmonary Tuberculosis. Among the 6 negatives, apart from Radiological lesion two had sputum positive for AFB. So the Mantoux test is a sensitive for pulmonary TB like Non diabetes patients.

Fig 2 : Radiological features of patients with Diabetes and pulmonary Tuberculosis.

Total : 26 Male : 20 Female : 6





G. Body mass Index :

TABLE-9.

Table 9 showing, BMI variation between DM with pulmonary TB and DM alone.

SUBJECTS	BMI
DM with pulmonary TB	18.92
DM alone.	23.67

It shows, Relatively underweight patients of Diabetes are more prone for pulmonary Tuberculosis infection

H.HbA1c VARIATION BETWEEN TB WITRH DM AND DIABETES ALONE;

TABLE-10.

Table 10 showing, HbA1c variation between DM with pulmonary TB and DM alone.

SUBJECTS	HbA1c in %
DM with pulmonary TB	8.01
DM alone.	6.47

HbA1c results shows, that diabetes with pulmonary TB had more than 8% compared with diabetes alone, it shows less than 8%.It indicate uncontrolled diabetes more prone for pulmonary TB

Table -11

Number of lesions in subjects with DM and Pul. Tuberculosis.

Side of Lesion	SEX		Total
	Male	Female	
Bilateral	4	2	6
Left	1	0	1
Right	15	4	19

Table -12

Table showing Blood sugar levels and Radiological Features Correlation.

S. No.	Sex	Bl. Glucose (mgs%)	No. Zone involved	Type of lesion
1.	M	321	2	® Consolidation
2.	M	210	1	® Infiltration
3.	M	465	4	B/L Infiltration
4.	M	300	2	® Consolidation
5.	M	193	1	® Infiltration
6.	F	261	1	® Infiltration
7.	M	317	2	® Fibrocavity
8.	M	400	6	B/L Infiltration
9.	M	210	1	® Infiltration
10.	F	364	6	B/L Infiltration
11	F	282	2	® Cavity
12	M	297	2	® Cavity
13	M	330	3	B/L fibrocavity
14	M	278	1	® Infiltration
15	M	312	2	® Consolidation
16	M	296	1	® Infiltration
17	M	323	3	® Fibrocavity
18	F	267	1	® Infiltration
19	M	292	2	® Consolidation
20	M	282	2	® Infiltration
21	F	305	2	® Consolidation
22	M	319	2	® Cavity
23	M	326	3	® Cavity
24	M	465	4	B/L Infiltration
25	F	390	4	B/L Infiltration
26	M	292	1	(L) Infiltration

Table -13

Showing Number of Zones Involved

No. of zones involved	Number of Subjects
One	8
Two	10
Three	3
Four	3
Five	-
Six	2

Table -14

Types of lesions and the pattern of distribution of subject with DM with Pulmonary TB.

Type of Lesion	Cases	Break up of Side of Lesion		
		Bilateral	Right	Left
Infiltration	14	5	8	1
Consolidation	5	-	5	-
Cavity	4	-	4	-
Fibrocavity	3	1	2	-

[7]All patients with tuberculosis were started 6 months ATT treatment according to the revised National Tuberculosis Control programme. The sputum positive tuberculosis patient were treated with DOTS Regimen, Containing 4 drug in the initial 2 months as intensive phase, are INH; Rifampicin; Ethambutol and pyrazinamide, and INH and Rifampicin for another 4 months as continuation phase. Diabetes was controlled with appropriate therapy (insulin) According to the continuous Bloodglucose monitoring. All sputum positive cases, subjected to sputum Examination for AFB after 2 months of intensive phase for monitoring of sputum conversion from positive to negative.

To monitor conversion and detect possible emergence of drug resistance, sputum smear and cultures should be obtained monthly or at least after 2^{nd}, 4^{th} and 6^{th} months of therapy. With INH and rifampicin containing regimens, sputum should convert to negative within 2 months. If smear and culture results continue to be positive, after 2 months of therapy, emerging drug resistance and non-compliance should be major concern. A new drug susceptibility test should be performed immediately.

Unless drug resistance is documented, the regimen in use should be continued carefully under direct observation. If drug resistance occurs at least 2 new drugs to which the organisms is sensitive should be added to the therapy and administered under direct observation. Additions of a single drug to therapy

should never be done since it will increase the risk of the rapid development of resistance to the new drugs. Bacteriological cultures and susceptibility tests should be performed at monthly intervals until the cultures become negative. Relapse of drug sensitive infection after adequate INH and Rifampicin containing treatment is very infrequent. For patients who have completed a standard regimen and had a satisfactory bacteriological response, follow up after completions of therapy is not necessary. In contrast, the patient with extensive disease, immunosuppressive patient, the persistence of radiological findings after therapy, or suspicious of poor patient compliance, are indications of prolonged follow up.

Table -15

Table showing interaction between severity of diabetes and Radiological lesions.

1. **Minimal Radiological lesion :**

   Single zone involved without affecting the other part.

2. **Moderate :**

   More than one zone involved or Bilateral Single Zone involvement, but less than four zone involvement.

3. **Far advanced :**

Bilateral Multiple Zone involved.

Radiological lesions	**Diabetes**		
	150-250 mild	**251-350 moderate**	**351 and above severe**
Minimal	3	5	-
Moderate	-	13	-
Far advanced	-	-	5

CONCLUSION

The reason for choosing this topic for my study is diabetes is so prevalence in INDIA and worldwide, tuberculosis is also more common in India, so far the relation between these two common diseases may be useful for controlling pulmonary tuberculosis and helpful for the healthy life of diabetic population.

The followings are conclusion drawn from the above study of patients with DIABETES MELLITUS and PULMONARY TUBERCULOSIS.

1. The prevalence of pulmonary Tuberculosis in diabetic patients is 26%

2. The male and female ratio of pulmonary tuberculosis in diabetic patients is 3.3:1.

3. The age distribution of patients suffering from both pulmonary tuberculosis and diabetes mellitus ranges from 30-79 years, more in the age group between 60 to 69 years.

4. Blood glucose is increased in combination of diabetes mellitus and pulmonary Tuberculosis compared with diabetes alone.

5. Out of 26 Subjects, 12 subjects were sputum positive for Acid Fast bacilli, 8 males and 4 females.

6. 20 subjects showed positive Mantoux, 6 were negatives. All positive Mantoux subjects, had pulmonary Tuberculosis. Among the 6 negatives, apart from Radiological lesion two had sputum positive for AFB.

7. Radiological studies showed subject had 6 Bilateral lesions. Regarding to sex 4 males and 2 female had bilateral lesion, that is, 15.38% males and 7.69% females had bilateral lesions.

8. Severity of Radiological lesion correlated well with the severity of DM.

9. Lower lobe involvement occurred in two patients, that is, 7.69%.

10. The insulin requirement is more (double) in patient with pulmonary tuberculosis than Diabetes alone.

11. Any diabetes patients showing sudden increase in insulin requirement is an indication to screen for pulmonary Tuberculosis.

12. Patient on oral Hypoglycemic agents need insulin Replacement.

13. Apart from 6 months course of Anti TB treatment, meticulous control of diabetes status is a must.

14. According to HbA1c results, uncontrolled diabetes has more prone for pulmonary tuberculosis.

15. Early screening and good control of Hyperglycemia are essential in preventing the onset of tuberculosis.

Chart - 1 : Diagram Showing Percentage of Diabetes with pulmonary tuberculosis against Diabetes alone.

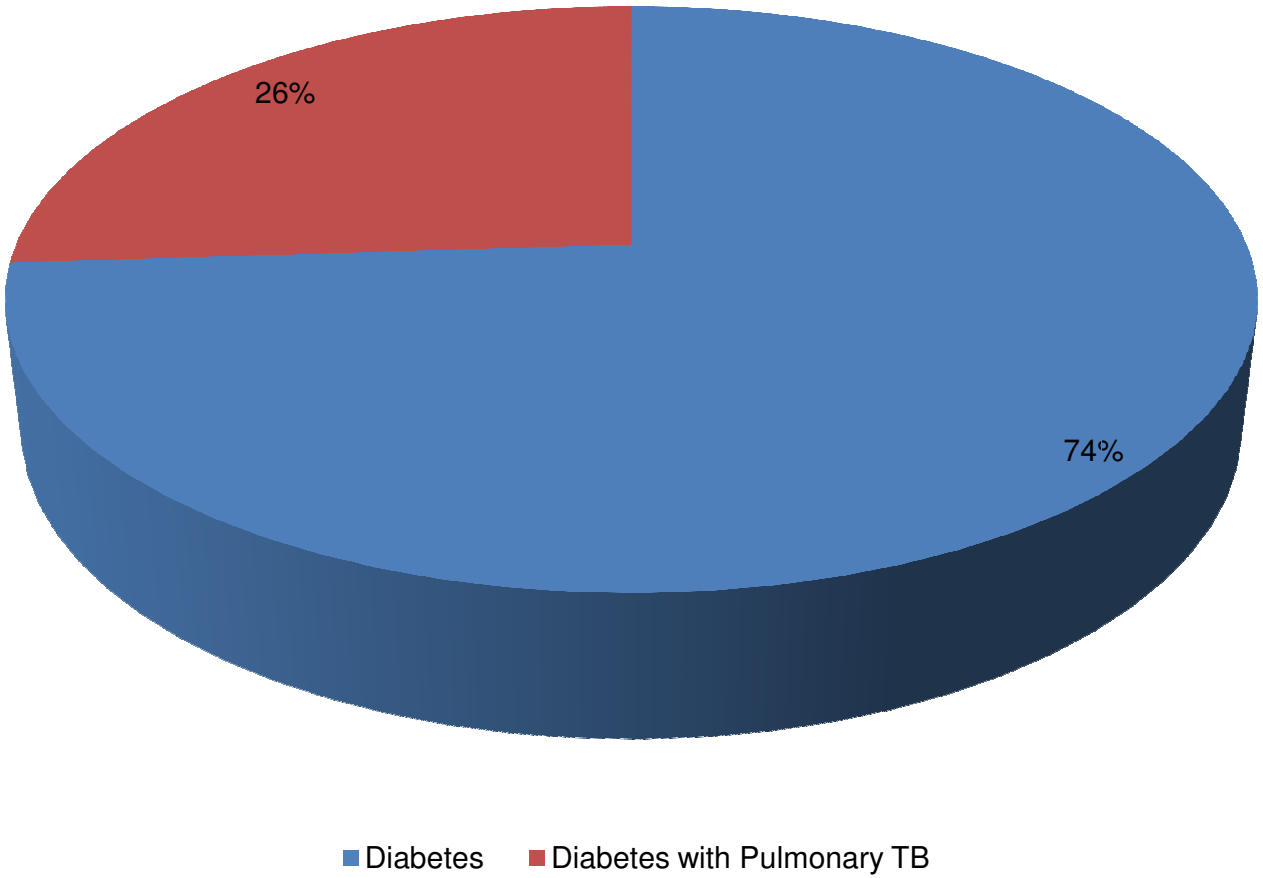
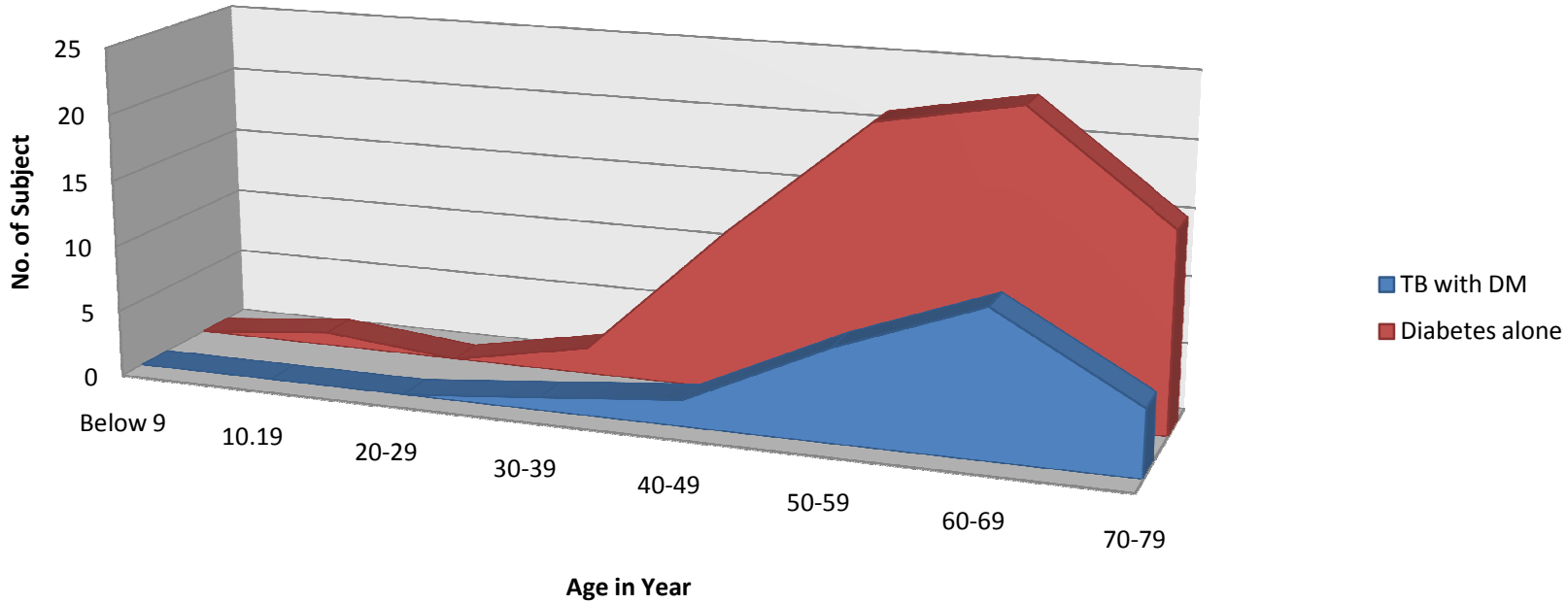


Chart - 2 : Age group (years) and Subjects with diabetes plus T.B. and Diabtest alone



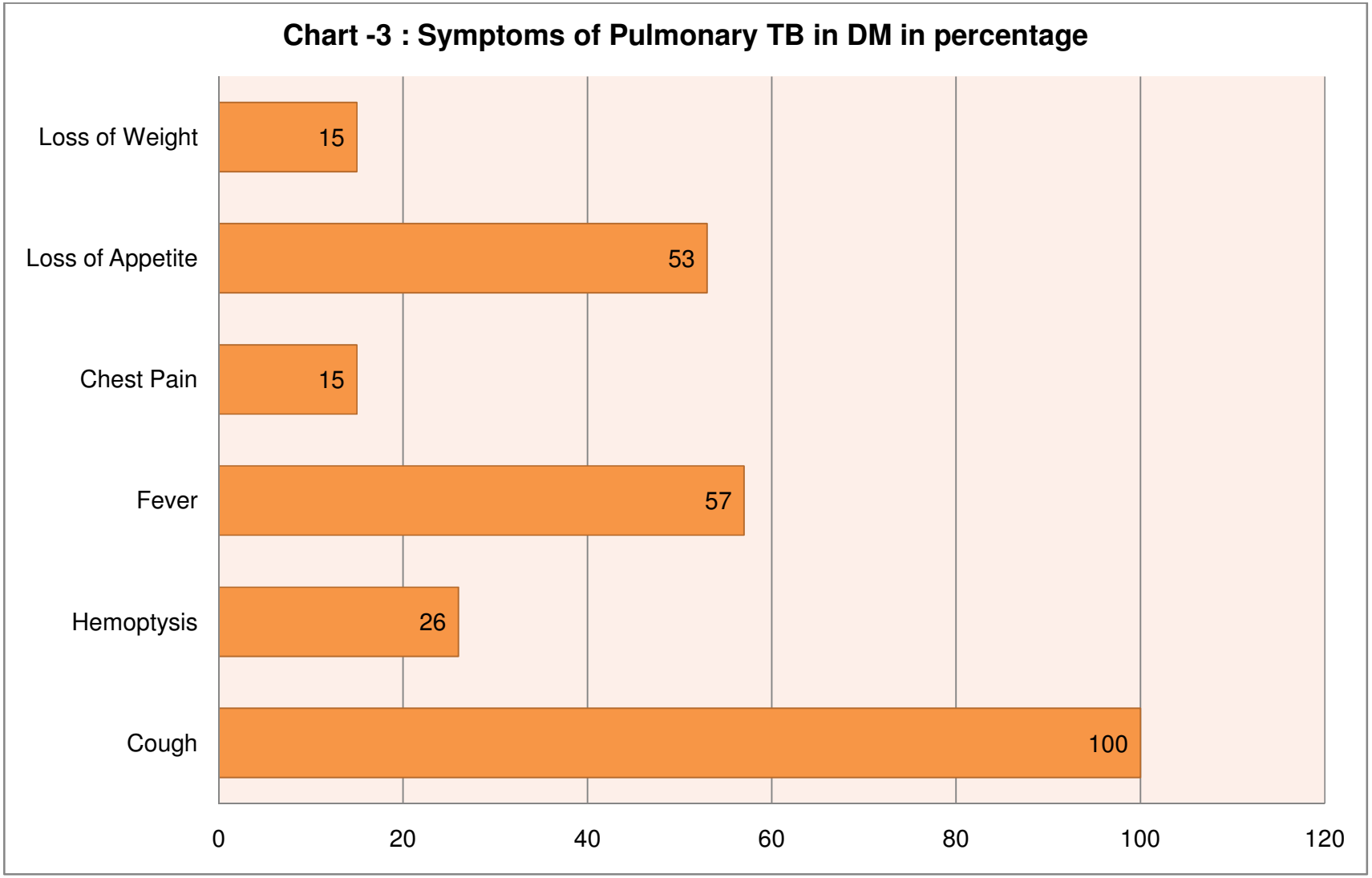


Chart -3 : Symptoms of Pulmonary TB in DM in percentage
Loss of Weight
15
Loss of Appetite
53
Chest Pain
15
Fever
57
Hemoptysis
26
Cough
100
0
20
40
60
80
100
120

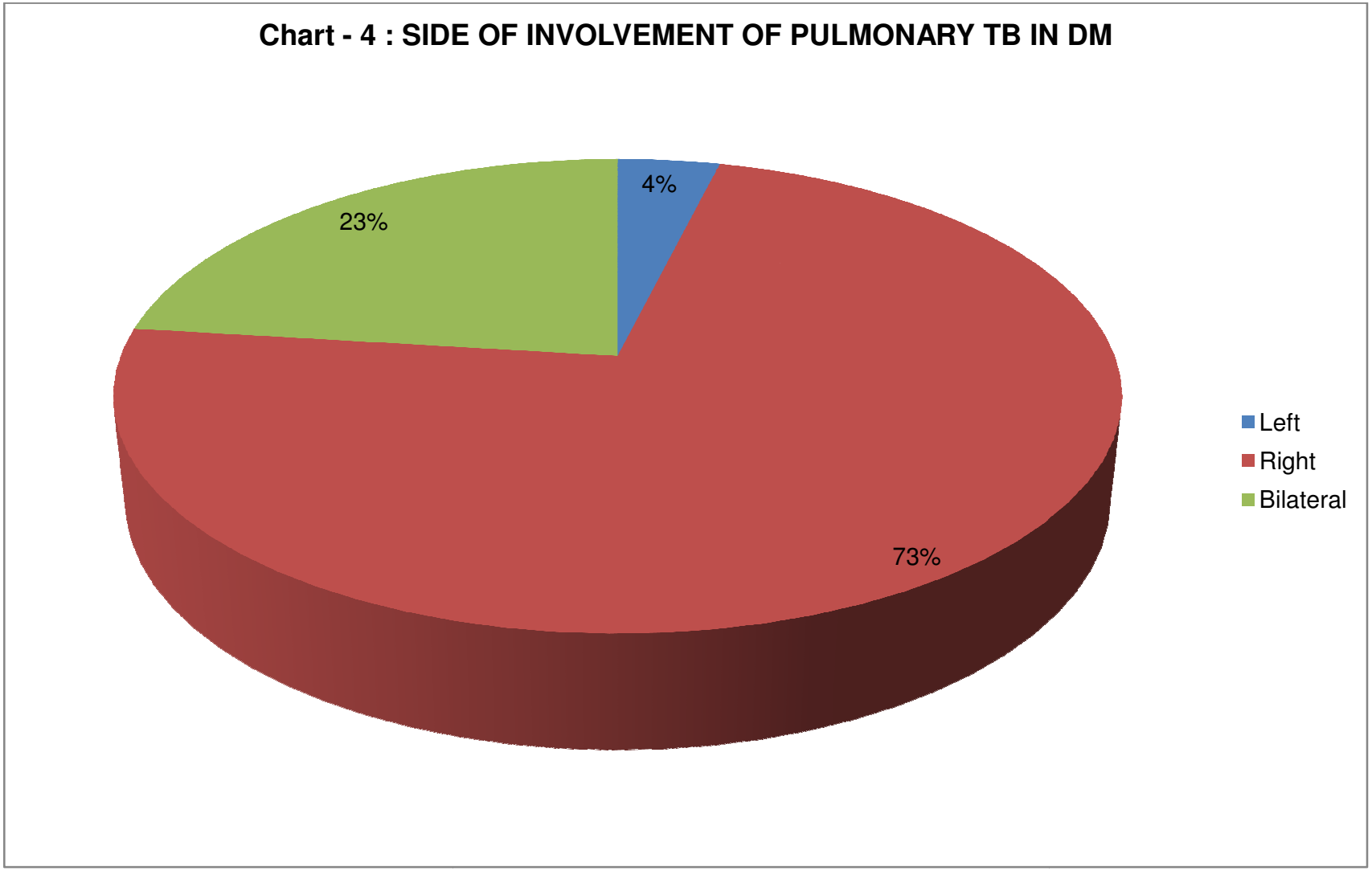


Chart - 4 : SIDE OF INVOLVEMENT OF PULMONARY TB IN DM
4%
23%
73%
Left
Right
Bilateral

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PROFORMA

1. Name :
2. Age :
3. Sex :
4. ID Number :
5. Address :

6. Diabetes : Y / N

   How many yrs :

   Type :

   Treatment :

7. Presenting Complaints :

   Cough –

   Hemoptysis –

   Fever –

   Chest Pain –

   Loss of weight –

   Others –

8. General Examination

Conscious :
Orientation :
Anemia :
Clubbing :
Cyanosis :
Pedal edema :
Lymphadenopathy ;

Height : Weight :

BMI :

PR : Rate / min
Regular / irregular
Vessel Wall thickening
Carotid bruit + / -

BP : mm Hg
RR : / min

9. CVS :
Apical Impulse :
Heart Sounds :
Murmurs :

10. CNS :

11. P/A :

12. RS :

Upper respiratory tract

Trachea

Chest deformities

Respiratory Movement

Vocal fremitus / resonance

Percussion

Auscultation

INVESTIGATIONS:

1. Urine R/E :

   Albumin

   Sugar

   Deposits

   Ketones

2. RFT :

   Urea

   Creatinine

   Electrolytes Na K HCO3

3. CBC :

TC	DC	ESR
Hb%	PCV	Platelets

4. FBS

   PPBS

5. ECG :

6. CXR PA View :

7. Mantoux :

8. Sputum AFB :

9. HbA1C :

10. CT Chest (if necessary) :

CONSENT FORM

1. I agree to participate in the study titled **"Study of current trends of prevalence and clinical spectrum of Pulmonary Tuberculosis in Diabetes Mellitus and its relationship with the glycemic status"**

2. I confirm that I have been told about this study in my mother tongue and have had the opportunity to ask questions.

3. I understand that my participation is voluntary and I may refuse to participate at any time without giving any reason and without affecting my benefits.

4. I agree not to restrict the use any data/results that arise from this study.

Name of the participant:

Signature/thumb print:

Witness: Investigator

S. No	Name of the Patient	Age	Sex	Rx Insulin	Cough with expectoration	Hemoptysis	Fever	Chest pain	Loss of appetite	Loss of weight	BMI	Mx	AFB	Hb A1c	Urine Sugar	Fasting blood sugar	PP blood Sugar.	TC	DC P,L,E	ESR	X-ray chest	CT chest	Duration of DM Yrs
1	Kulanthaiyesu	56	M	15	+	-	+	+	-	-	27	Neg	Neg	6.9	+	119	212	6700	70202	10/22	-	-	6
2	Razul Baig	48	M	20	+	-	-	-	-	-	26	Neg	Neg	6.5	-	118	186	7500	65352	5/7	-	-	2
3	Ramalingam	47	M	OHA	+	-	+	-	-	-	21	+ve	Neg	8.1	++	250	392	7650	70282	9/18	® consolidation	® consolidation	8
4	Abbas ali	63	M	OHA	+	-	-	-	-	-	28	Neg	Neg	6.4	-	150	187	5800	65341	8/20	-	-	3
5	Vijaya	46	F	OHA	+	-	-	-	-	-	29	Neg	Neg	6.9	+	100	233	4700	72062	10/22	-	-	5
6	Kadalaiyammal	62	F	0HA	+	-	-	--	+	-	20	Neg	Neg	7.1	+	170	221	6800	76222	8/32	-	-	6
7	Andalammal	56	F	OHA	+	-	-	-	-	-	22	Neg	Neg	6.5	-	40	170	6200	65341	5/12	-	-	8
8	Saroja	58	F	OHA	+	-	+	-	-	-	26	Neg	Neg	6.9	+	160	212	6800	55422	6/14	-	-	10
9	Nagarajan	60	M	OHA	+	-	+	-	-	-	20	Neg	Neg	7.9	+	180	240	4600	65350	18/32	® Upper Zone infiltration	-	11
10	Ravi	51	M	45	+	+	+	-	+	-	19	+ve	+ve	9.1	+++	419	512	5600	71281	12/23	B/L infiltration	-	8
11	Anandan	49	M	40	+	-	+	-	+	+	15	+ve	+ve	8.0	++	220	380	7900	69301	12/25	® Upper& Middle zone consolidation		4
12	Balaraman	55	M	OHA	+	-	+	+	-	-	21	+ve	Neg	7.2	+	168	218	6400	55450	19/35	® Upper zone infiltration	-	15
13	Mutharasi	53	F	OHA	+	-	-	-	+	-	18	Neg	Neg	7.5	++	180	342	6100	72262	6/14	® upper zone infiltration	-	8
14	Gobinath	75	M	35	+	-	-	-	+	-	16	+ve	+ve	8.1	+++	249	396	8600	60364	6/14	® Upper& Middle zone fibrocavity	-	6
15	Valliammal	66	F	OHA	+	-	+	-	+	-	24	Neg	Neg	7.3	++	180	348	9800	80200	25/55	-	-	5

S. No	Name of the Patient	Age	Sex	Rx Insulin	Cough with expectoration	Hemoptysis	Fever	Chest pain	Loss of appetite	Loss of weight	BMI	Mx	AFB	Hb A1c	Urine Sugar	Fasting blood sugar	PP blood Sugar.	TC	DC P,L,E	ESR	X-ray chest	CT chest	Duration of DM Yrs
16	Joney Basha	62	M	OHA	+	-	-	-	+	-	26	Neg	Neg	6.8	-	130	196	6300	82180	5/12	-	-	15
17	Sagunthala	48	F	OHA	+	-	-	-	-	-	26	Neg	Neg	6.7	-	126	190	6100	80200	10/22	-	-	6
18	Kuppusamy	68	M	OHA	+	-	+	+	-	-	28	Neg	Neg	6.8	+	113	220	5900	60373	5/12	-	-	8
19	Elumalai	58	M	OHA	+	-	-	-	-	-	20	Neg	Neg	6.5	-	107	160	8400	50482	26/40	-	-	2
20	Dilli	56	M	45	+	+	-	-	-	-	20	+ve	+Ve	9.2	+++	322	477	4900	58384	26/40	B/L diffuse infiltration	-	6
21	Elumalai	49	M	OHA	+	-	+	+	-	-	29	Neg	Neg	7.2	++	212	280	8000	70300	10/16	-	-	3
22	Munusamy	59	M	OHA	+	-	+	-	+	-	18	Neg	Neg	7.2	+++	180	240	6200	71281	15/35	® upper zone infiltration	-	8
23	Sardha Beevi	62	F	45	+	-	+	-	+	-	19	+Ve	+Ve	8.1	+++	312	415	5600	71290	10/25	B/L diffuse infiltration	-	10
24	Parvathi	52	F	OHA	+	-	-	-	-	-	19	Neg	Neg	6.6	+	120	191	4800	68311	8/024	-	-	8
25	Chellammal	64	F	OHA	+	-	-	-	+	+	29	Neg	Neg	6.5	+	102	212	6200	68302	5/12	-	-	2
26	Layal	62	M	OHA	+	-	-	-	-	-	22	Neg	Neg	6.5	+	128	216	5000	63371	6/14	-	-	4
27	Jamila	47	F	OHA	+	-	-	--	-	--	20	Neg	Neg	6.2	+	87	221	6100	52480	6/15	-	-	3
28	Lakshmi	64	F	36	+	+	-	-	+	-	16	+ve	+ve	7.6	++	244	320	8400	69274	10/26	® Upper & middle zone cavity	-	7
29	Jothilakshmi	18	F	15	+	-	-	+	-	-	27	Neg	Neg	6.4	+	129	212	8900	80200	5/12	-	-	2
30	Kuppammal	63	F	OHA	+	-	-	-	+	-	21	Neg	Neg	6.3	+	124	208	6000	80200	5/20	-	-	18
31	Surya Narayanan	68	M	OHA	+	-	+	-	-	-	21	Neg	Neg	7.1	+	215	256	8500	60400	5/12	-	-	16

S. No	Name of the Patient	Age	Sex	Rx Insulin	Cough with expectoration	Hemoptysis	Fever	Chest pain	Loss of appetite	Loss of weight	BMI	Mx	AFB	Hb A1c	Urine Sugar	Fasting blood sugar	PP blood Sugur.	TC	DC P,L,E	ESR	X-ray chest	CT chest	Duration of DM Yrs
32	Kalaivanan	63	M	OHA	+	-	-	+	-	-	20	+ve	Neg	7.8	++	225	368	6900	68320	6/15	® Upper & Middle zone cavity	-	7
33	Mannakatti	63	M	OHA	+	-	-	-	-	-	20	Neg	Neg	6.8	+	138	212	6000	63343	5/12	-	-	2
34	Janardhanan	60	M	45	+	+	-	-	-	-	20	+ve	+ve	8.2	++	280	380	1100	55450	10/32	B/L Fibrocavity	B/;L Fibrocavity	20
35	Saravanan	39	M	OHA	+	-	+	-	-	-	22	Neg	Neg	7.4	+	239	317	6900	82153	3/7	® Upper Zone infiltration	-	70
36	Jameed Basha	53	M	OHA	+	-	-	-	-	-	27	Neg	Neg	7.0	+	126	250	6600	60400	6/15	-	-	6
37	Ravi Selvam	38	M	24	+	-	-	-	-	-	22	Neg	Neg	6.4	-	106	178	4200	55414	7/15	-	-	2
38	Minnala	51	F	30	+	-	+	-	-	-	26	Neg	Neg	6.8	+	180	235	6700	75250	4/10	-	-	4
39	Lakshmi	56	F	OHA	+	-	-	-	-	-	26	Neg	Neg	6.4	-	128	180	8400	69254	16/24	-	-	8
40	Jayaraman	58	M	OHA	+	-	-	-	-	-	27	Neg	Neg	6.5	-	127	198	7100	72280	6/24	-	-	4
41	Dhamodharan	56	M	OHA	+	-	+	+	+	-	20	Neg	Neg	6.6	+	159	231	4500	68302	18/28	-	-	2
42	Mani	51	M	OHA	+	-	-	-	-	-	29	Neg	Neg	6.4	+	160	214	6000	70300	6/40	-	-	8
43	Mutharasan	57	M	OHA	+	-	+	-	-	-	17	+ve	Neg	8.0	++	282	342	11300	76240	18/40	® upper & middle zone consolidation	-	7
44	Bebi John	71	F	OHA	+	-	-	-	-	-	22	Neg	Neg	6.2	+	104	215	7100	75250	6/15	-	-	4
45	Mohan	47	M	OHA	+	-	-	-	-	-	25	Neg	Neg	6.4	-	123	189	6900	61354	10/19	-	-	1
46	Basker	65	M	36	+	-	+	-	-	-	23	+ve	+ve	7.8	++	225	368	7500	70300	2/15	® upper zone infiltration	-	5

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47	Krishnaveni	46	F	OHA	+	-	+	-	-	-	20	Neg	Neg	6.4	+	126	184	6700	55405	5/20	-	-	6
48	Venkadakondaiya	67	M	OHA	+	-	-	-	+	-	21	Neg	Neg	6.6	+	160	212	5800	72280	10/27	-	-	2
49	Thangavelu	59	M	OHA	+	-	-	+	-	-	18	Neg	Neg	6.4	-	120	186	6800	652510	10/21	-	-	10
50	Samboornam	58	F	OHA	+	-	+	-	-	-	20	Neg	Neg	6.5	-	128	198	6400	62371	5/12	-	-	4
51	Paul mary	56	F	OHA	+	-	-	-	-	-	27	Neg	Neg	6.4	+	196	212	6700	70282	10/22	-	-	6
52	Parus	64	M	38	+	+	+	-	+	+	15	+ve	+ve	8.1	++	249	396	7900	69301	12/23	® fibrocavity	-	6
53	Govindammal	68	F	20	+	-	-	-	-	-	18	Neg	Neg	6.2	-	112	186	7500	63352	7/15	-	-	2
54	Ambathika	47	F	OHA	+	-	+	+	-	-	21	Neg	Neg	7.2	+	212	295	7650	70282	9/18	-	-	8
55	Devan	54	M	OHA	+	-	+	-	-	-	25	Neg	Neg	6.4	-	125	190	6800	65341	9/20	-	-	3
56	Rajendran	57	M	OHA	+	-	-	-	-	-	19	Neg	Neg	6.1	+	100	233	4700	60355	10/22	-	-	5
57	Munusamy	67	M	OHA	+	-	-	-	-	-	28	Neg	Neg	6.4	-	158	245	6100	72262	19/35	-	-	3
58	Ramamoorthy	62	M	OHA	+	-	-	-	-	-	20	Neg	Neg	7.2	++	210	301	6800	76222	18/33	-	-	6
59	Muthu	60	M	OHA	+	-	-	-	--	-	23	Neg	Neg	6.4	-	90	170	6200	65341	5/12	-	-	2
60	Amala	52	F	OHA	+	-	+	-	-	-	20	Neg	Neg	6.6	+	160	212	6800	55422	5/12	-	-	2
61	Priyamvada	50	F	OHA	+	-	+	-	-	-	20	Neg	Neg	7.4	++	214	320	4200	65350	6/14	® upper zone infiltration	-	11
62	Indira	60	F	OHA	+	-	+	+	-	-	29	Neg	Neg	6.5	+	160	220	5600	71281	8/32	-	-	2
63	Kumar	65	M	OHA	+	-	+	-	-	-	21	+ve	Neg	7.5	++	250	335	6400	55450	12/25	® upper& middle zone consolidation	-	15

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64	Jaya	70	F	16	+	-	-	-	-	-	26	Neg	Neg	6.4	-	132	197	8600	64324	6/14	-	-	4
65	Devaraj	38	M	OHA	+	-	-	-	-	-	20	Neg	Neg	6.6	+	180	248	9800	80200	4/10	-	-	6
66	Mumtaj	64	F	OHA	+	-	-	-	-	-	26	Neg	Neg	6.2	-	130	196	6100	80200	5/12	-	-	6
67	Susaimani	76	M	OHA	+	-	+	-	-	-	26	Neg	Neg	6.1	-	126	190	6100	80200	5/12	-	-	6
68	Yesodha	72	F	24	+	-	-	-	-	-	28	Neg	Neg	6.2	+	113	220	5900	60373	10/22	-	-	6
69	Mallika	53	F	OHA	+	-	-	-	-	-	20	Neg	Neg	6.0	-	107	156	8400	50482	5/12	-	-	2
70	Vijaya	58	F	OHA	+	-	-	-	-	-	19	Neg	Neg	6.8	+	212	218	8000	70300	9/9	-	-	3
71	Krishnan	72	M	OHA	+	-	-	-	-	-	20	Neg	Neg	7.6	++	200	364	4500	58324	26/40	® upper& middle zone infiltration	-	6
72	Kasthuri	65	F	45	+	+	+	+	+	-	18	+Ve	+ve	8.1	++	239	371	6200	72280	15/36	® upper& middle zone infiltration	® upper& middle zone infiltration	8
73	Deen Mohamed	69	M	OHA	+	-	-	-	-	-	19	+ve	Neg	8.3	++	288	350	5600	71290	10/25	® upper& middle zone cavity	-	10
74	Kalaiselvi	46	F	OHA	+	-	-	-	-	-	19	Neg	Neg	6.1	-	120	191	4800	68311	8/24	-	-	20
75	Maragatham	78	F	OHA	-	-	-	-	-	-	29	Neg	Neg	6.2	+	102	212	6200	68522	5/12	-	-	10
76	Sulochana	47	F	OHA	+	-	+	+	-		21	Neg	Neg	6.2	_	128	216	5000	63371	6/14	-	-	4
77	Kamrudeen	75	M	OHA	+	-	-	-	-		20	Neg	Neg	6.3	+	87	221	6100	52481	6/15	-	-	3
78	Chandran	77	M	OHA	+	-	-	-	-	-	16	Neg	+ve	8.1	++	290	362	8400	69274	10/26	® sided cavity	-	7

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79	Annabakiyan	72	M	30	+	-	+	-	-	-	Neg	23	Neg	6.2	+	128	216	5000	63371	6/14	-	-	8
80	Veerakumar	70	M	OHA	+	-	+	-	-	-	Neg	24	Neg	6.8	+	215	256	8500	60400	5/12	-	-	6
81	Narayanan	74	M	OHA	+	-	-	-	-	-	Neg	21	Neg	6.8	+	215	256	8500	60400	5/12	-	-	6
82	Vishalashi	71	F	45	-	-	-	-	-	+	Neg	20	+ve	9.2	+++	419	512	6900	68320	6/15	B/L Upper &Middle zone infiltration	-	7
83	Senthamarai	61	F	OHA	+	-	-	-	-	-	Neg	20	Neg	6.3	+	138	212	6000	63343	5/12	-	-	2
84	Rajesh	73	M	OHA	+	-	-	-	-	-	Neg	21	Neg	9.0	+++	360	420	11000	55450	10/32	B/L infiltration	-	12
85	Eswaran	46	M	OHA	+	-	-	-	-	-	Neg	22	Neg	6.4	+	168	218	6900	82153	3/7	-	-	2
86	Riaz	58	M	OHA	+	-	-	-	-	-	Neg	22	Neg	6.6	+	126	250	6600	60400	6/15	-	-	4
87	Malliga	62	F	OHA	+	-	-	-	-	-	Neg	31	Neg	6.4	+	106	178	4200	55414	7/15	-	-	2
88	Victoria	74	F	18	+	-	-	-	-	-	Neg	21	Neg	6.4	_	180	235	6700	7525	4/10	-	-	3
89	Chandhran	63	M	OHA	+	-	-	-	-	-	Neg	26	Neg	6.4	-	128	180	8400	69256	16/24	-	-	4
90	Savithri	75	F	OHA	+	-	-	-	-	-	+ve	27	Neg	6.1	-	127	198	7100	72280	6/24	-	-	4
91	Chitra	70	F	OHA	+	-	-	-	-	-	Neg	20	Neg	6.2	+	150	231	11500	63352	18/28	-	-	4
92	Shanthi	48	F	OHA	+	-	+	-	-	-	Neg	19	Neg	6.3	+	160	214	8000	70300	18/40	-	-	5
93	Ramesh	42	M	36	+	-	+	-	+	-	Neg	17	Neg	7.8	++	250	335	11300	70300	18/40	L-upper zone infiltration	L-upper zone infiltration	7
94	Alima	66	F	OHA	+	-	-	-	-	-	Neg	32	Neg	6.2	+	105	215	7100	76240	6/14	-	-	8
95	Krishnan	54	M	OHA	+	-	-	+	-	-	Neg	25	Neg	6.0	-	123	189	6900	75250	6/15	-	-	1

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96	Umamaheswari	62	F	OHA	+	-	-	-	-	-	Neg	24	Neg	6.4	++	142	224	7500	61354	10/19	-	-	2
97	Raman	65	M	OHA	+	-	-	-	-	-	Neg	28	Neg	6.2	+	160	212	5800	70300	2/15	-	-	2
98	Somasundharam	72	M	15	+	-	-	-	-	-	Neg	26	Neg	6.4	+	140	286	6800	55405	5/10	-	-	6
99	Rajendran	76	M	OHA	+	-	-	-	-	-	Neg	22	Neg	6.2	-	128	198	6400	72280	10/27	-	-	3
100	Krishnakumar	70	M	OHA	+	-	-	-	-	-	Neg	22	Neg	6.1	-	126	184	670	622510	10/21	-	-	6